Immunol., vol. 159, pages 3100-3103, 1997) suggested through animal experiment of multiple sclerosis using lpr and gld mouse, which are genetically deficient of the Fas and the Fas ligand, respectively, that the apoptosis mediated by the Fas/Fas ligand is involved in the multiple sclerosis. In the meanwhile, Eileen, A. et al. (J. Clin. Invest., vol. 98, pages 1602-1612, 1996) and Suzana, M. et al. (J. Exp. Med., vol. 186, pages 507-515, 1997) suggested through animal experiment of the multiple sclerosis using the same lpr and gld mouse that the apoptosis mediated by the Fas/Fas ligand is not involved in the multiple sclerosis. In other words, the relationship between the pathology of the multiple sclerosis and the apoptosis mediated by the Fas/Fas ligand system is still unknown and differently conceived depending on the investigator. In addition, efficiency of the drug delivery to brain tissue is generally low, and it is utterly unknown whether the drug which suppresses the apoptosis by the Fas/Fas ligand administered to the body can suppress the Fas/Fas ligand-mediated apoptosis in the brain tissue, and it is also unknown whether the results will be the same as those obtained in the mouse genetically deficient of the Fas or the Fas ligand.--

Please replace the paragraph beginning on page 16, line 23 with the following rewritten paragraph:

--The anti-Fas ligand antibody and the anti-Fas antibody used in the present invention may be prepared by known process, for example, by the process described in International Patent

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Application Publication No. WO95/13293 and International Patent Application Publication No. WO97/02290. These publications are herein incorporated by reference.--

Please replace the paragraph beginning on page 19, line 11 with the following rewritten paragraph:

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--Another preferable Fas derivative is the Fas having a deletion in its N terminal. Among these, Fas derivatives, the shFas(nd29)-Fc and the shFas(nd29)-hinge (International Patent Application Publication No. WO 97/42319) coded in plasmids (pM1304 and pM1317) included in the E. coli which were originally deposited in March 14, 1996 in National Institute of Bioscience and Human Technology, Agency of Industrial Science and Technology (1-3, Higashi 1-chome, Tsukuba-shi, Ibaraki-ken, Japan) (Accession Nos. P-15514 and P-15515) and transferred from the original deposition to the international deposition on March 6, 1997 (Accession No. FERM BP-5854 and Accession No. FERM BP-5855) are derivatives including the extracellular domain of the known human Fas from which N terminal sequence of from 1st to 29th amino acid has been deleted, and these highly active derivatives are preferable examples of the effective component for the preventive and therapeutic agent of autoimmune demyelinating diseases of the present invention. This publication is herein incorporated by reference.--

Please replace the paragraph beginning at page 31, line 6 with